

FIG. 1

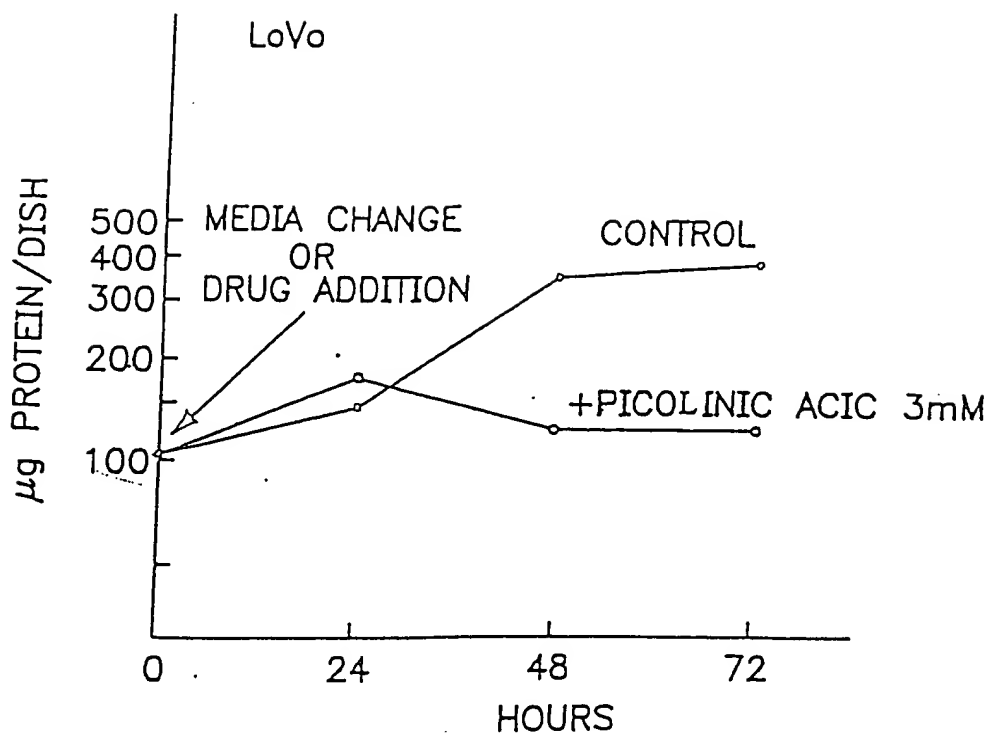


FIG. 2

FIG. 3A

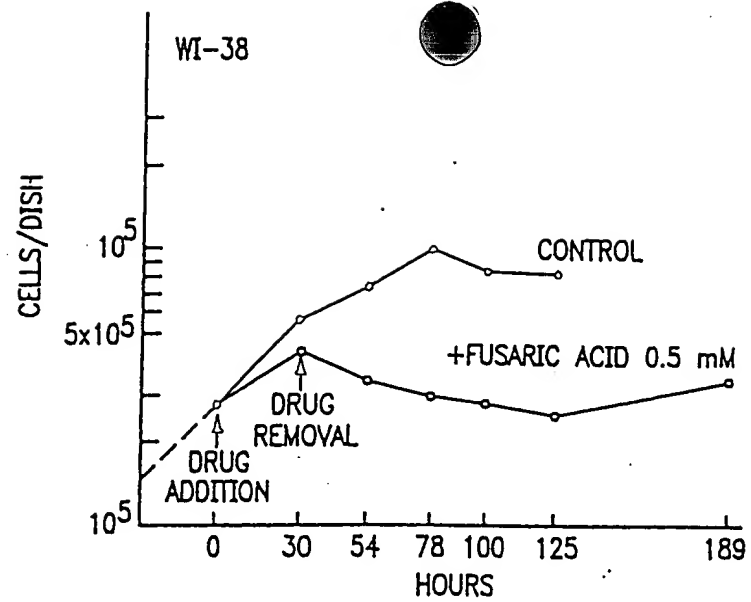


FIG. 3B

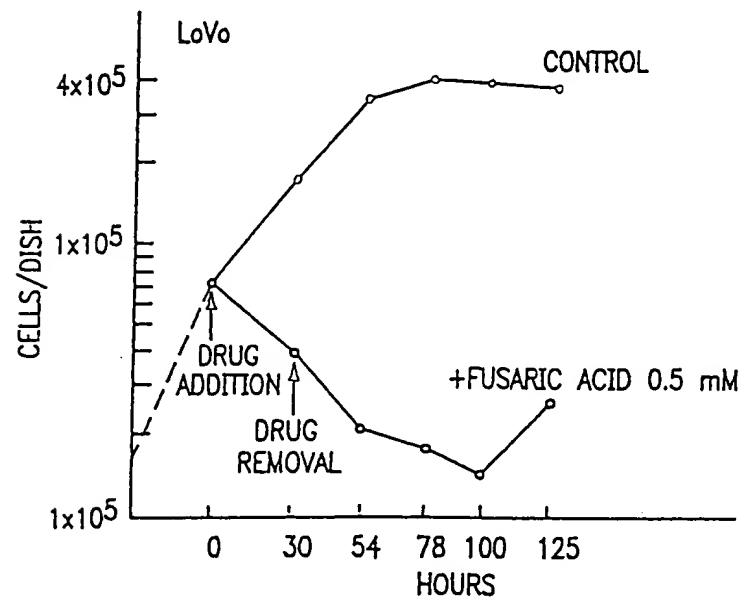


FIG. 3C

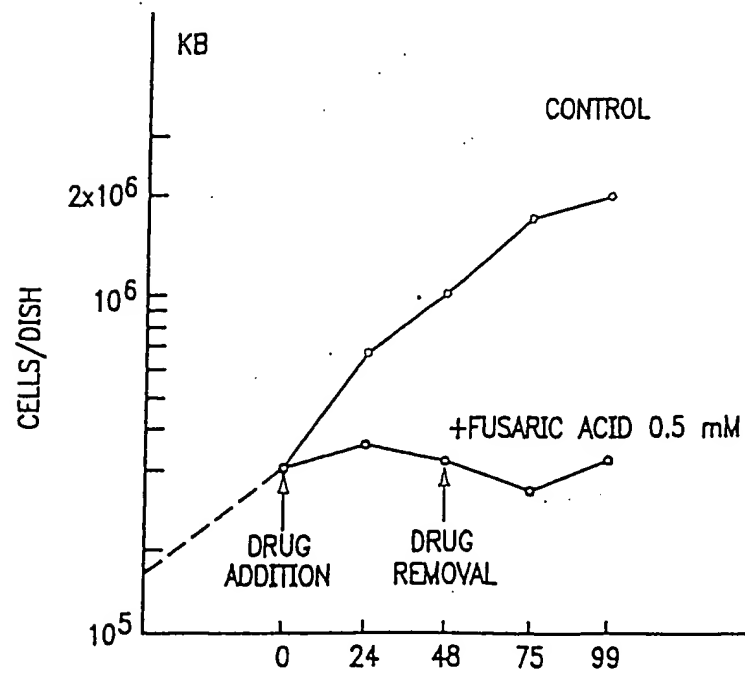


FIG. 4A



FIG. 4B



FIG. 4C

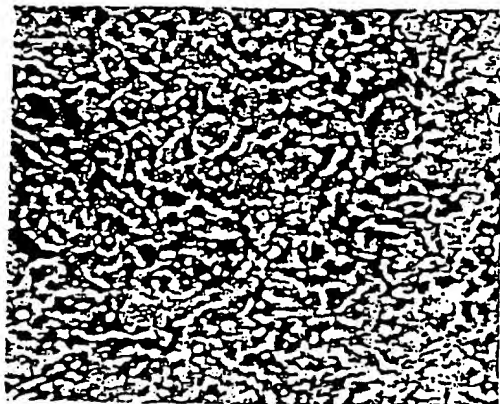


FIG. 4D

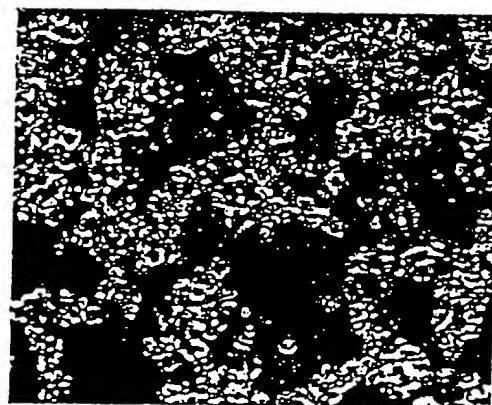


FIG. 5A



FIG. 5B

MODULATION OF APOPTOSIS BY INTRACELLULAR
CONCENTRATIONS OF ZINC

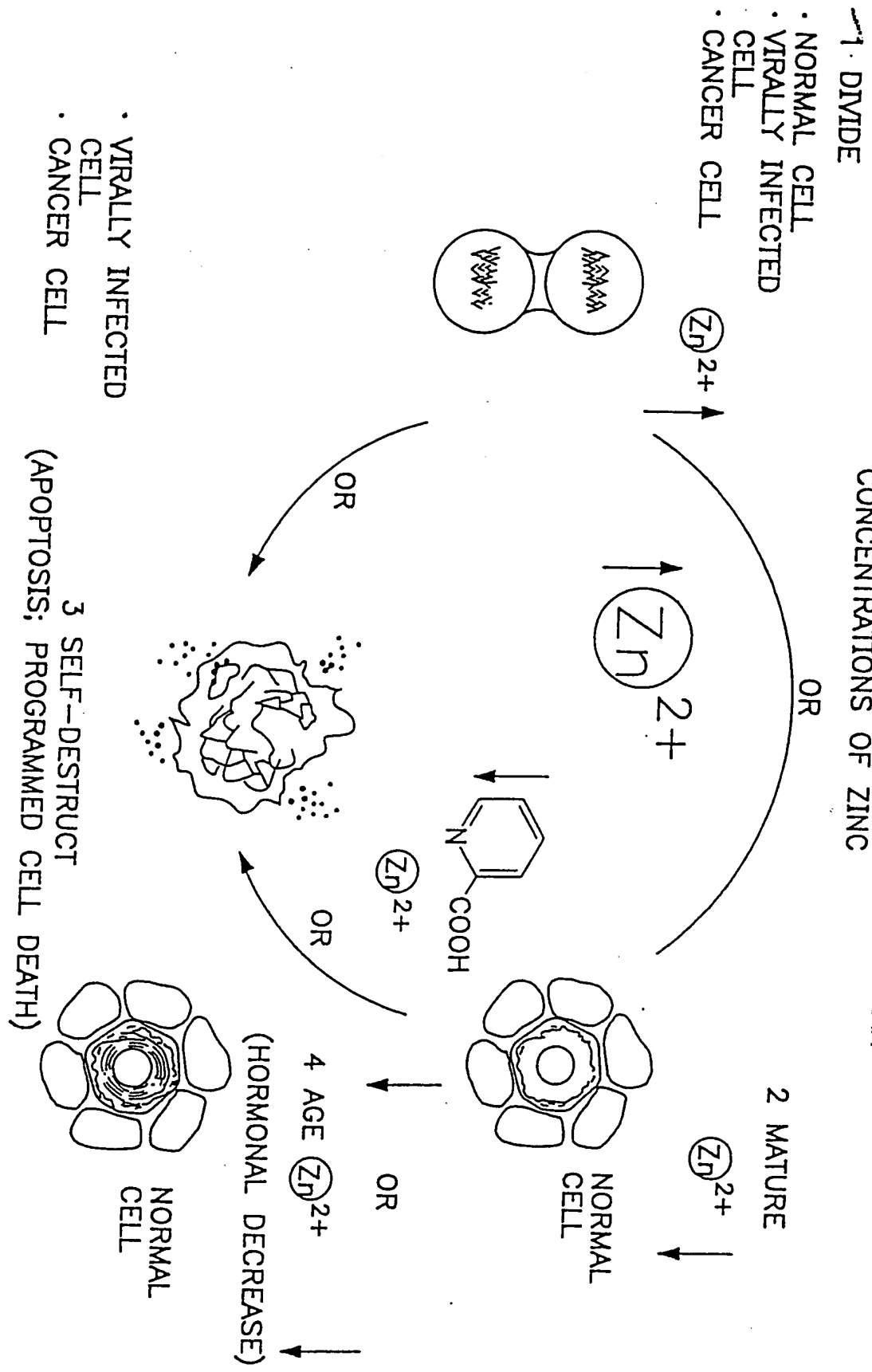


FIG. 6

The diagram illustrates the HIV infection cycle and the role of zinc finger proteins. At the top, a 'VIRION' is shown with its characteristic surface spikes. The cycle proceeds through several stages: 1. Entry into the cell, where the viral RNA is released. 2. Reverse transcription, where the RNA is converted into 'PROVIRUS (DOUBLE-STRANDED) DNA'. 3. Integration into the host genome, where the provirus DNA is inserted into the 'NUCLEUS'. This step involves the 'MPS (S27)' protein, which is shown binding to the DNA. A 'POOL OF Zn^{2+} ' is indicated as a cofactor for this process. 4. Replication and packaging, where the provirus is transcribed into new viral RNA. This step involves the 'p7' protein and another 'POOL OF Zn^{2+} '. 5. Budding, where new virions are released from the cell. The diagram also shows a 'RIBOSOME MPS/S27' and a 'PACKAGING STEP' box. The overall process is labeled 'HIV INFECTION AND ZINC FINGER PROTEINS INVOLVED IN VIRAL HYPERCYCLES'.

PACKAGING STEP

HIV INFECTION, ZINC FINGER PROTEINS AND ANTIRETROVIRAL ACTIVITY OF PICOLINIC ACID AND DERIVATIVES

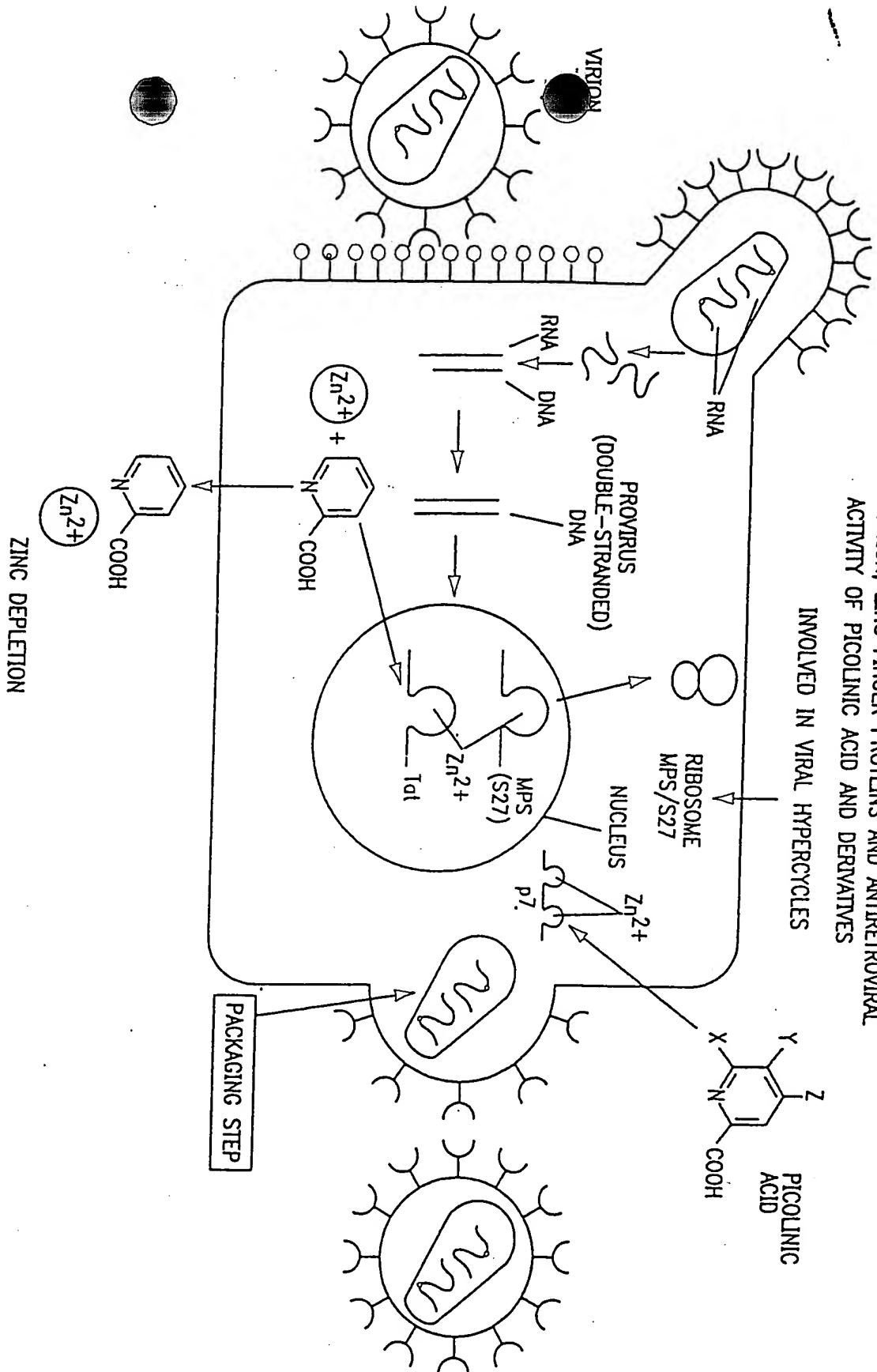
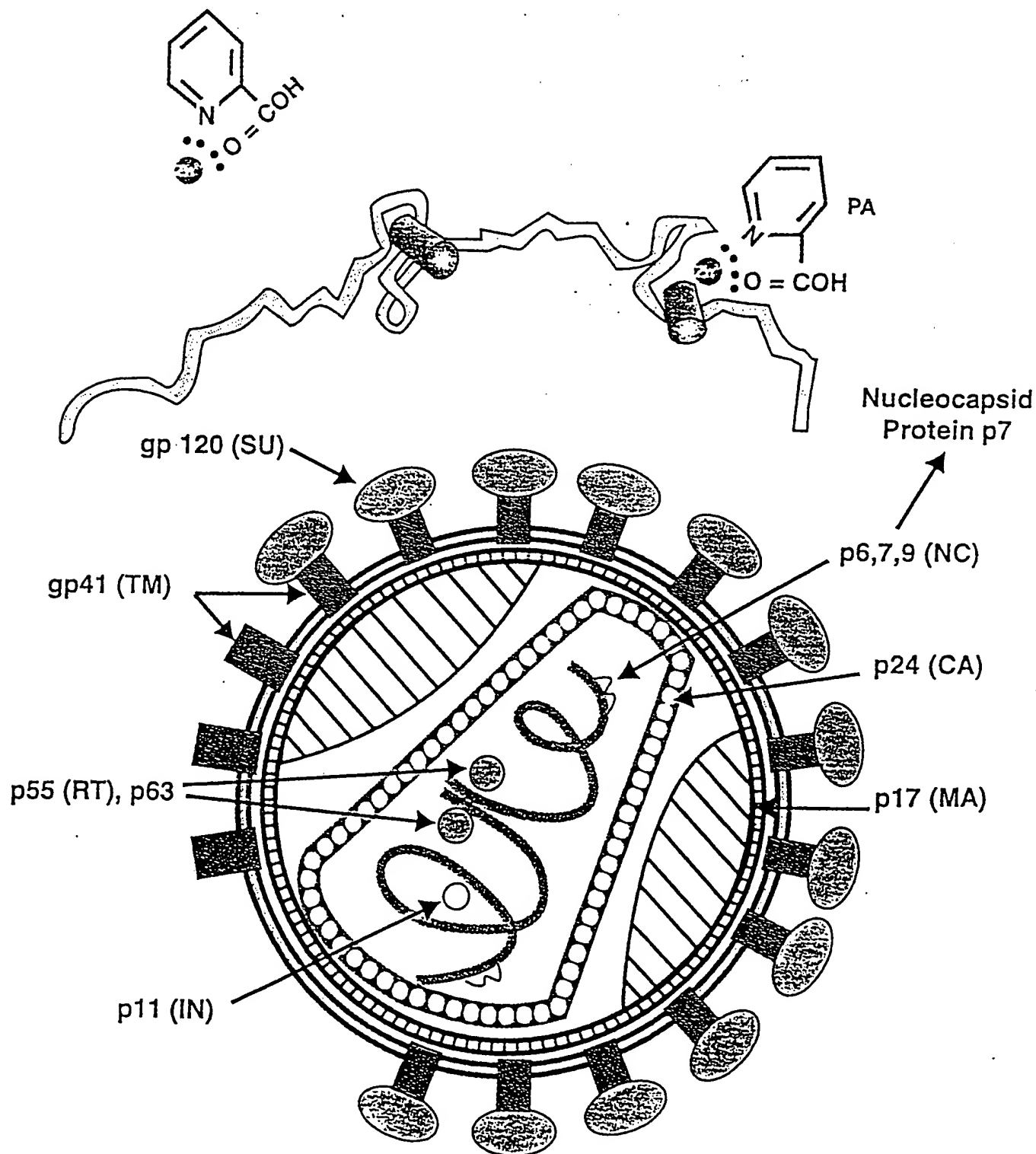
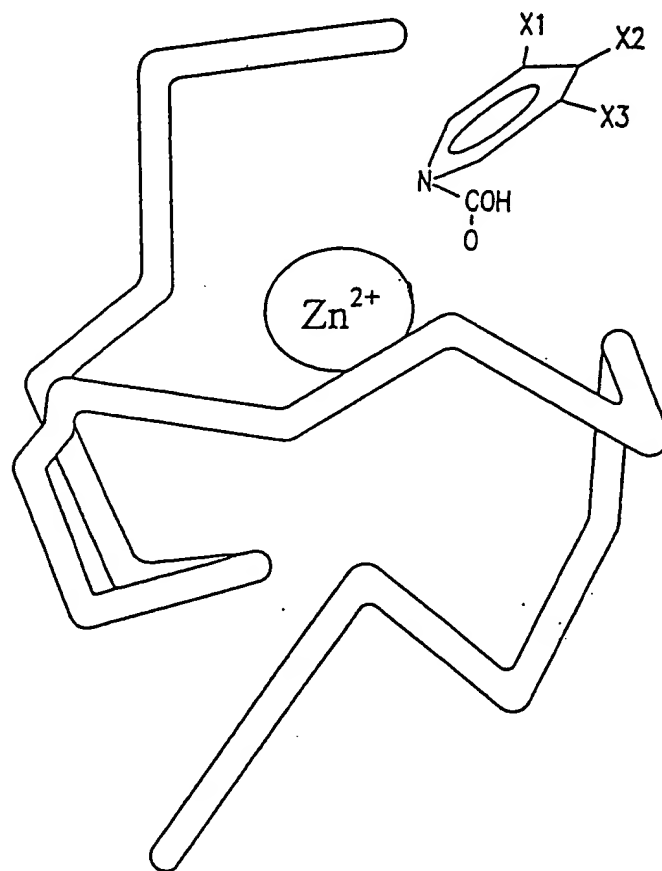


FIG. 8

Disruption of Zinc Finger Binding Domains of Retroviral Proteins by PA



THE WIDE SPECTRUM ANTIVIRAL ACTIVITY
OF PA-X_n ARE DUE TO DISRUPTION OF THE
ZINC FINGER BINDING DOMAINS
OF RETROVIRAL PROTEINS



PA-X_n ABOLISH THE ZINC FINGER PROTEINS
ABILITY TO BIND RNA

FIG. 10

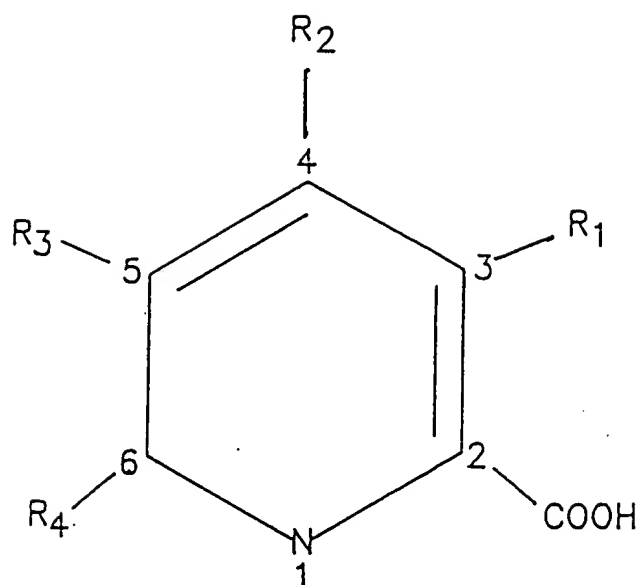
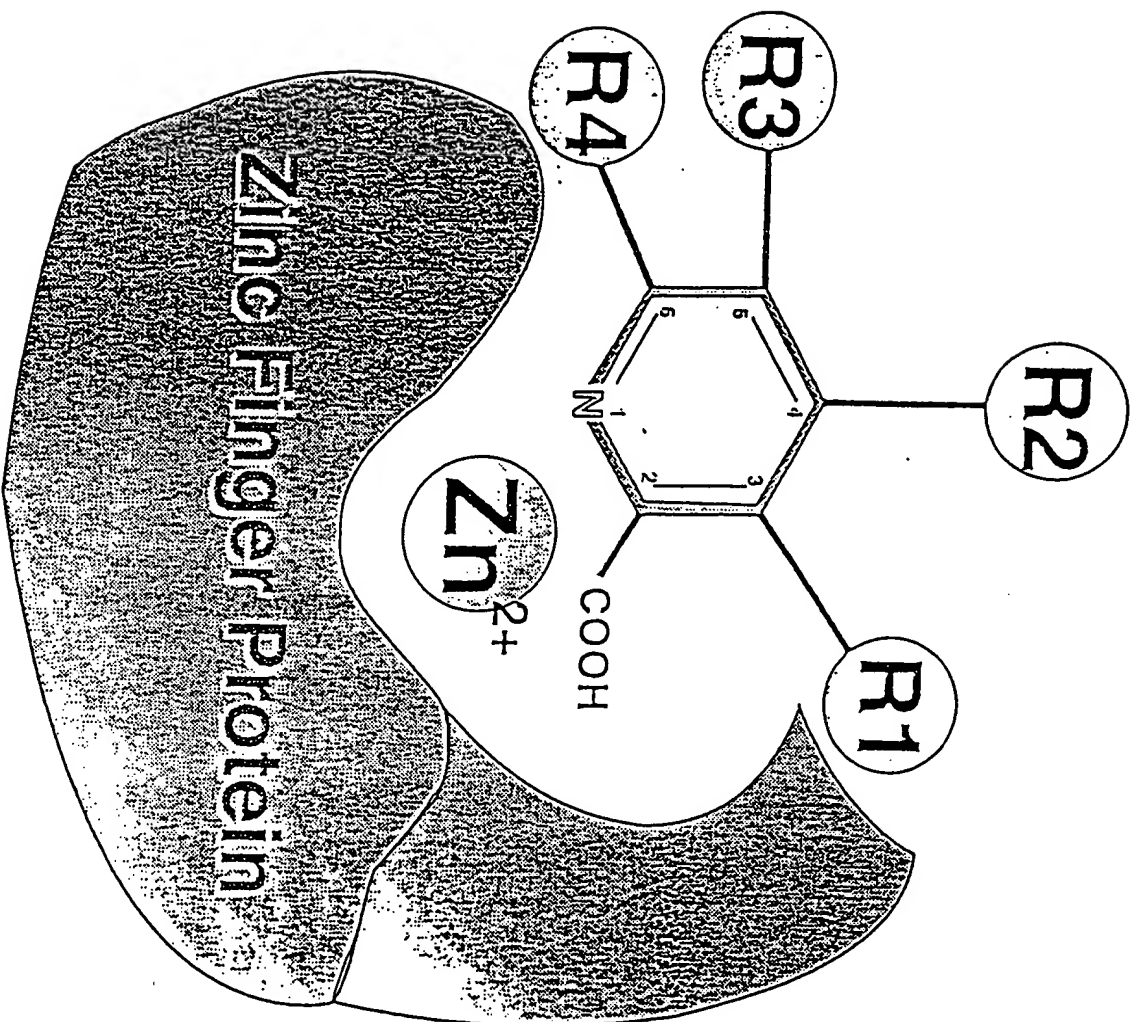


FIG. II



VIRAL INFECTION, RIBOSOMAL PROTEINS AND HSPTS

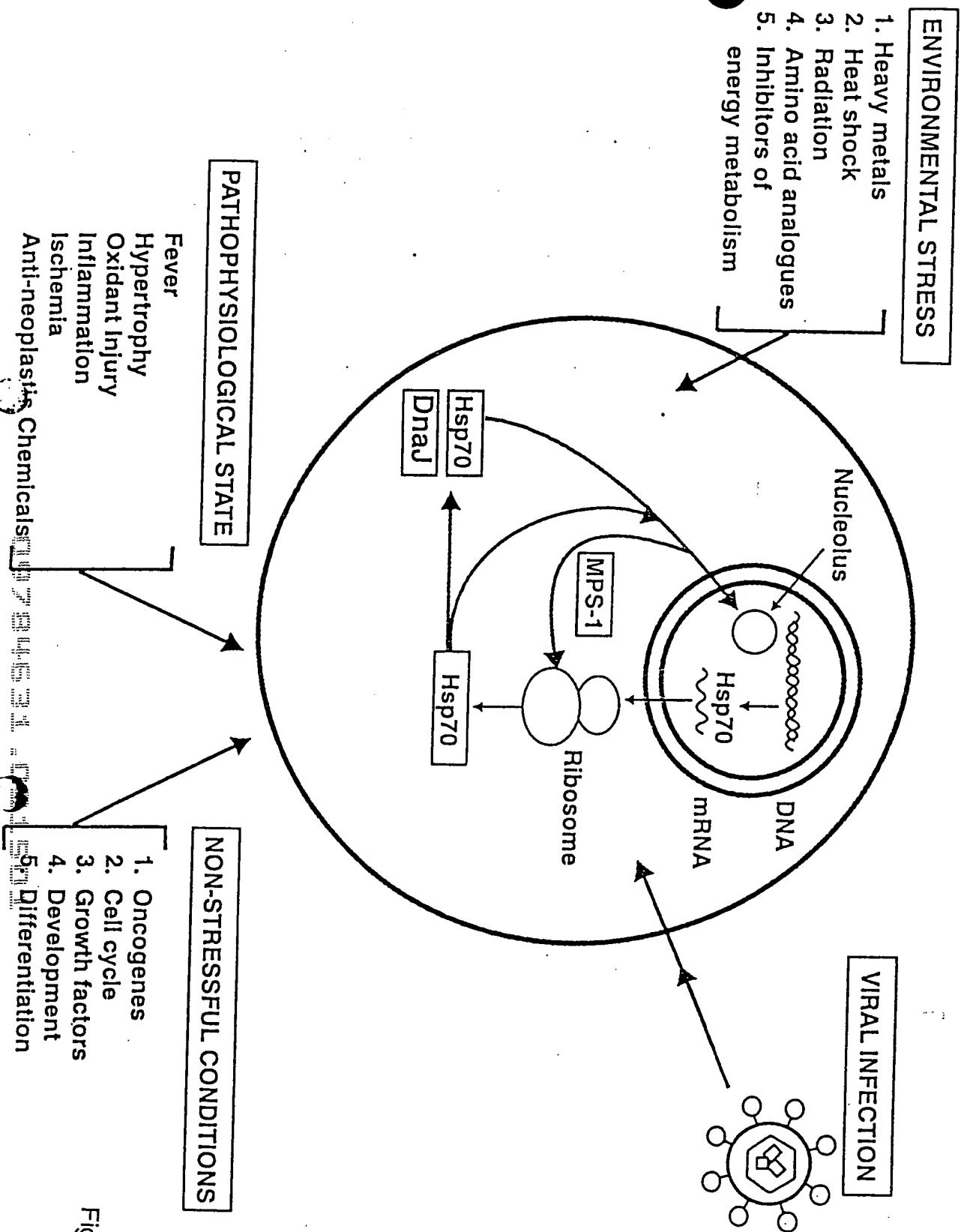


Figure 13